

Supplement Material

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Do The Health Benefits Of Cycling Outweigh The Risks?

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Supplement Material, Table 1 Human controlled exposure studies of traffic-related particulate matter air pollution

Study population	Exposure	Main findings	Ref.
Fifteen healthy young subjects	1 hour with intermittent exercise to 300 µg/m ³ diesel exhaust	No changes in lung function directly after exposure but marked systemic and pulmonary inflammation six hours after exposure, e.g. 3-4 fold increase in neutrophils in bronchial tissue and 1.5 fold increase of neutrophils in blood.	Salvi, 1999; Salvi 2000
Ten young healthy subjects	2 hour at rest to 200 µg/m ³ diesel exhaust	No changes in lung function, HR, BP and inflammation markers in blood. Significant increase in exhaled CO indicating oxidative stress (+50%), neutrophils (+28%) and other markers of airway inflammation in sputum 4 hr after exposure.	Nightingale, 2000
Healthy subjects and mild asthmatics	2 hour to 108 µg/m ³ diesel exhaust particles	No change in lung function. Increase in airway resistance (4.1% in healthy subjects and 6.5% in asthmatics). Increase in airway inflammation in healthy subjects (+40% for neutrophils), not found in asthmatics. Increase in cytokines (IL-6, IL-8) in healthy subjects and IL-10 in asthmatics.	Stenfors, 2004
Thirty health young subjects	1 hour with intermittent moderate exercise to 300 µg/m ³ diesel exhaust	No change in blood inflammatory markers, 34% reduction of fibrinolytic capacity (tissue plasminogen activator) and reduction of response to vasodilator (smaller increase in forearm blood flow, vasomotor function). At 24-hour vasomotor function changes persisted and systemic inflammation was found (IL-6, TNF-α).	Mills, 2005; Törnqvist, 2007
Twenty men with a prior myocardial infarction	1 hour with intermittent exercise to 300 µg/m ³ diesel exhaust	Asymptomatic increase in indicators of myocardial ischemia (doubling of ST-segment depression and ischemic burden) and 35% reduction of fibrinolytic capacity possibly resulting in thrombosis. No change in inflammation markers and vasomotor function.	Mills, 2007
Twenty-three healthy subjects	2 hour at rest to 147 ± 27 µg/m ³ PM _{2.5} (CAP) + 121 ± 3 ppb ozone. Mean OC 25 ± 12 µg/m ³	Increase of diastolic blood pressure of ~10% and no changes in HR. Associations especially with OC (traffic) and not with total PM _{2.5} .	Urch, 2005
Fifteen healthy volunteers	2 hour with intermittent exercise to 100 µg/m ³ diesel exhaust particles	Increased bronchial but not alveolar inflammation indicated by increased neutrophil and mast cell counts and inflammation markers IL-8 and MPO in bronchial wash. Increase in anti-oxidants urate and glutathione in alveolar lavage. All health effects are for 18 hr post-exposure.	Behndig, 2006
Twelve healthy and twelve asthmatic subjects	2 hour with intermittent exercise to on average 174µg/m ³ PM _{2.5} (CAP)	No changes in lung function or routine hematologic measurements. Both groups showed decreases of columnar cells in postexposure induced sputum, slight changes in mediators of blood coagulability and systemic inflammation, and modest increases in parasympathetic stimulation of heart rate variability. Systolic blood pressure decreased in asthmatics and increased in healthy subjects. Cardiovascular (but not respiratory) symptoms increased slightly in both groups.	Gong, 2003
Twenty CHD and twenty healthy subjects	1 hour to 50 µg/m ³ carbon particles and 200 ppb SO ₂	No effect was found on systemic inflammation and heart rate variability.	Routledge, 2006

Seventeen healthy and fourteen asthmatic subjects	2 hour with intermittent exercise to on average 145,000 p/cm ³ ultrafine particles (CAP)	Small decrements of lung function, oxygen saturation in blood and heart rate variability in both healthy and asthmatic volunteers	Gong, 2008
Fourteen young healthy subjects	1 hour with intermittent exercise to 300 µg/m ³ diesel exhaust particles followed by 0.2 ppm ozone 5 hrs later	Significant increase in airway inflammation as indicated by neutrophils, macrophages and eosinophils in bronchial wash, but not alveolar lavage. The study documents interaction between diesel and ozone exposure.	Bosson, 2008
Twenty healthy subjects	2 hour to 350 µg/m ³ diesel exhaust 1 hour to 350 µg/m ³ diesel exhaust	Increased platelet activation and thrombus formation, which may lead to cardiac events such as MI in patients	Lucking, 2008
Ten healthy young subjects	1 hour in rest to 300 µg/m ³ diesel exhaust	Physiological changes observed in electroencephalography (EEG), indicative of a general cortisol stress response.	Crüts, 2008
Twelve CHD and twelve healthy subjects	2 hour intermittent moderate exercise to 190 ± 37 µg/m ³ PM _{2.5} (CAP) and 99,400 p/cm ³ UFP	No effect on vasomotor function, BP, HR, fibrinolytic function and systemic inflammation, though small increases of platelets and monocytes were found. About four-fold increase in 8-isoprostane in exhaled breath condensate (oxidative stress) in 8 healthy subjects. Likely due to composition of PM (90% sea salt)	Mills, 2008

Note: in all studies post-pre exposure effects on health were compared with filtered air exposure of the same exercise and duration.

BHR, bronchial reactivity test; HR, heart rate; BP, blood pressure; CAP, concentrated ambient particles; OC, organic carbon fraction of PM_{2.5}; CHD, coronary heart disease; UFP, ultrafine particles; MI, myocardial infarction.

Supplement Material, Table 2a

Calculation of the potential mortality impact of cycling compared to car driving, under various assumptions of the exposure in the car, the increase in inhaled dose due to cycling, and the relative risk to calculate the mortality impact for PM_{2.5}

Activity	Minute ventilation (l/min.) ^a	Duration (hr/day)	Concentration PM _{2.5} (µg/m ³) ^b	Inhaled dose (µg/day) ^c	Total dose (µg/day) ^d	Ratio of total dose bicycle/car	Mean PM _{2.5} (µg/m ³) ^e	Equivalent change in PM _{2.5} (µg/m ³) ^f	RR mortality, equal toxicity ^g	RR mortality, traffic 5x more toxic
Half hour commute										
Sleep	5.0	8.0	20.0	48.0						
Rest	10.0	15.5	20.0	186.0						
Car	10.0	0.5	30.0	9.0	243					
Cycle	22.0	0.5	25.9	17.1	251	1.03	20.2	0.7	1.004	1.020
Sleep	5.0	8.0	20.0	48.0						
Rest	10.0	15.5	20.0	186.0						
Car	10.0	0.5	40.0	12.0	246					
Cycle	22.0	0.5	34.5	22.8	257	1.04	20.4	0.9	1.005	1.026
One hour commute										
Sleep	5.0	8.0	20.0	48.0						
Rest	10.0	15.0	20.0	180.0						
Car	10.0	1.0	30.0	18.0	246					
Cycle	22.0	1.0	25.9	34.1	262	1.07	20.4	1.3	1.008	1.040
Sleep	5.0	8.0	20.0	48.0						
Rest	10.0	15.0	20.0	180.0						
Car	10.0	1.0	40.0	24.0	252					
Cycle	22.0	1.0	34.5	45.5	274	1.09	20.8	1.8	1.010	1.053

Supplement Material, Table 2b

Calculation of the potential mortality impact of cycling compared to car driving, under various assumptions of the exposure in the car, the increase in inhaled dose due to cycling, and the relative risk to calculate the mortality impact for BS

Activity	Minute ventilation (l/min.) ^a	Duration (hr/day)	Concentration BS (µg/m ³) ^b	Inhaled dose (µg/day) ^c	Total dose (µg/day) ^d	Ratio of total dose bicycle/car	Mean BS (µg/m ³) ^e	Equivalent change in BS (µg/m ³) ^f	RR mortality, equal toxicity ^g	RR mortality, traffic 5x more toxic
Half hour commute										
Sleep	5.0	8.0	10.0	24.0						
Rest	10.0	15.5	10.0	93.0						
Car	10.0	0.5	20.0	6.0	123					
Cycle	22.0	0.5	12.1	8.0	125	1.02	10.2	0.2	1.001	1.004
Sleep	5.0	8.0	10.0	24.0						
Rest	10.0	15.5	10.0	93.0						
Car	10.0	0.5	30.0	9.0	126					
Cycle	22.0	0.5	18.2	12.0	129	1.02	10.4	0.2	1.001	1.006
One hour commute										
Sleep	5.0	8.0	10.0	24.0						
Rest	10.0	15.0	10.0	90.0						
Car	10.0	1.0	20.0	12.0	126					
Cycle	22.0	1.0	12.1	16.1	130	1.03	10.4	0.3	1.002	1.008
Sleep	5.0	8.0	10.0	24.0						
Rest	10.0	15.0	10.0	90.0						
Car	10.0	1.0	30.0	18.0	132					
Cycle	22.0	1.0	18.2	24.0	138	1.05	10.8	0.5	1.002	1.012

N.B.: Values are rounded.

- ^a Typical values for minute ventilation at sleep and rest assumed as 5 and 10 l/min (0.3 m³/h and 0.6 m³/h respectively). Minute ventilation for car drivers is assumed to be equivalent to that at rest 10 l/min (0.6 m³/h). Minute ventilation for cyclists is assumed to be 2.2 times the estimate for drivers (22L/min or 1.32 m³/h) based on information reported in van Wijnen et al. 1995 (minute ventilation of cyclists 2.3 times that of drivers) and Zuurbier et al. 2009 (minute ventilation of cyclists 2.1 times that of drivers).
- ^b Concentrations of PM_{2.5} and BS during sleep and rest are assumed to be equivalent to typical European urban background values (Putaud et al. 2010; Schaap and van de Gon, 2007) of 20µg/m³ and 10µg/m³, respectively. Concentrations during driving are assumed to be either 1.5 times or two times the background concentration for PM_{2.5} (scenario 1: 30, scenario 2: 40µg/m³) and 2 times or 3 times the background concentration for BS (scenario 1: 20, scenario 2: 30µg/m³) based on Zuurbier et al. 2010. Concentrations of PM_{2.5} and BS during cycling are assumed to be 0.862 and 0.606 times those estimated for drivers based on information summarized in Table 1 (scenario 1: 25.9µg/m³ and 12.1µg/m³ for PM_{2.5} and BS, respectively; scenario 2: 34.5µg/m³ and 18.2µg/m³ for PM_{2.5} and BS, respectively).
- ^c Inhaled dose (µg/day) = minute ventilation (m³/h) * duration (h/day) * concentration (µg/m³)
- ^d Total dose (µg/day) = Inhaled dose during sleep + rest + driving or cycling.
- ^e Time-weighted average PM_{2.5} or BS exposure over a 24-hour period for drivers (baseline situation).
- ^f Calculated as the fractional difference of the total dose for cycling versus car driving multiplied by the time weighted average PM_{2.5} concentration. For example, assuming a half hour commute under exposure scenario 1, the equivalent change in PM_{2.5} = [(251µg/day / 243µg/day) - 1] * 20.2µg/m³ = 0.7µg/m³, meaning that the added exposure in cyclists is equivalent to breathing an average PM_{2.5} concentration that is 0.7µg/m³ higher than the average concentration to which drivers are exposed.
- ^g RR, Relative Risks, estimated as: EXP[LN(1.06)*(equivalent PM_{2.5} change /10)], where 1.06 is the average adjusted relative risk of all cause mortality for a 10 µg/m³ change in PM_{2.5} concentration derived from the American Cancer Society study (Pope et al. 2002) and as EXP[LN(1.05)*(equivalent BS change /10)] where 1.05 is the average adjusted relative risk of natural cause mortality (i.e., all deaths excluding accidents and murders, ICD-9 > 800) for a 10 µg/m³ change in BS based on Beelen et al. 2008.
- ^h Estimates assuming that traffic exposures are 5 times more toxic than background exposures are estimated as EXP[(5*(LN(1.06))*(equivalent PM_{2.5} change /10)] for PM_{2.5} and as EXP[(5*(LN(1.05))*(equivalent BS change /10)] for BS.

Supplement Material, Table 3

Injuries and fatalities plus hospital admissions per age category per billion passenger kilometres by bicycle and by car within urban areas in the Netherlands (van Boggelen 2005).

Age	Injuries			Fatalities and hospital admissions		
	Bicycle	car	Risk ratio	Bicycle	car	Risk ratio
0-11	545	80	6,8	115	12	9,5
12-17	1164	349	3,3	199	72	2,8
18-24	1019	903	1,1	198	197	1,0
25-29	826	434	1,9	150	86	1,7
30-39	614	267	2,3	118	51	2,3
40-49	642	196	3,3	149	33	4,4
50-59	780	173	4,5	213	33	6,4
60-74	1018	204	5,0	329	52	6,3
75+	2567	449	5,7	1077	147	7,4
Total	882	280	3,2	205	56	3,7

Supplement material, Table 4 Mortality rates for being involved in a fatal traffic accident per billion kilometers traveled in the Netherlands, excluding motorways (European Commission, 1999).

Age category	Bicycle	Car
12-14	16.8	-
15-17	18.2	-
18-24	7.7	33.5
25-29	8.2	17.0
30-39	7.0	9.7
40-49	9.2	9.7
50-59	17.2	5.9
60-64	32.1	10.4
> 64	79.1	39.9
Total ^a	21.0	20.8

^a The average total risk is biased against cyclists because two (less experienced, less cautious) age groups which do not exist among motorists are taken into consideration. Non-weighted rates for the 18-64 yr old are 13.6 and 14.4 for cyclists and car drivers respectively. The rates include the risk that car drivers present to other road users (pedestrians, cyclists)

Supplement Material, Table 5 Analysis of life years gained /lost from shifting to bicycle use for a 7.5 km distance travelled per age group

Stressor	Age category	Baseline mortality rate^a	Mean Relative risk	Gain in life years ^a	Loss or gain in days / months per person*
Air pollution	18-39	238	1.03	-4153	-3 days
	40-64	1932	1.03	-26 019	-19 days
	65+	22 660	1.03	-83 788	-2 months
Traffic accidents	18-39	238	Age 18-29: 0.996	-806	-0.6 days
			Age 30-39: 1.009		
			Age 40-49: 1.010		
			Age 50-59: 1.005		
	40-64	1932	Age 60-64: 1.005	-4731	-3 days
			Age 65-69: 1.004		
			Age 70-79: 1.010		
Physical activity	65+	22 660	Age 80+: 1.003	-14 532	-11 days
	18-39	238	0.70	41 580	1 month
	40-64	1932	0.70	263 517	6 months
	65+	22 660	0.70	1 062 527	2 years

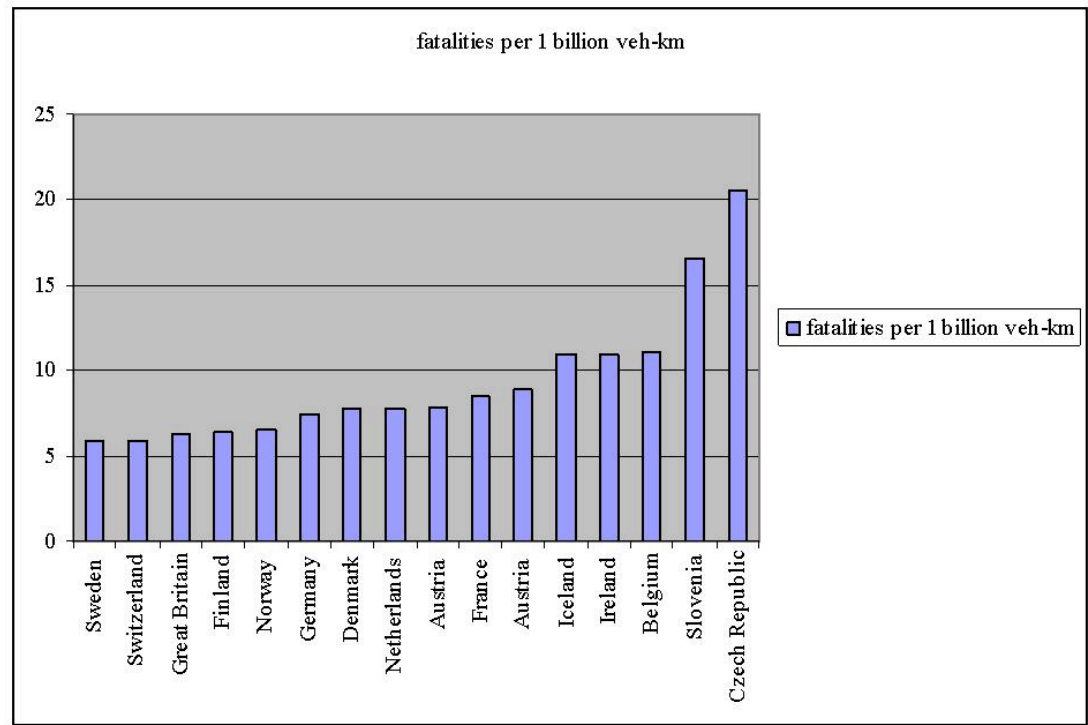
Values are rounded. Estimates are presented for the mean RRs in table 7. Values can be compared to the central estimate in table 6 in the main text. ^a applied to 500,000 subjects with different age categories with standard life table calculations (Miller and Hurley 2003). A minus sign implies losses

**Supplement Material, Table 6 Percentage of trips taken by walking and cycling in European countries and USA
(Bassett et al., 2008).**

Country	Year	Walking	Cycling
Austria	2005	21	4
Belgium	1999	16	8
Denmark	2003	16	15
Finland	2005	22	9
France	1994	19	3
Germany	2002	23	9
Latvia	2003	30	5
Netherlands	2006	22	25
Norway	2001	22	4
UK	2006	24	2
USA	2001	9	1
Sweden	2006	23	9

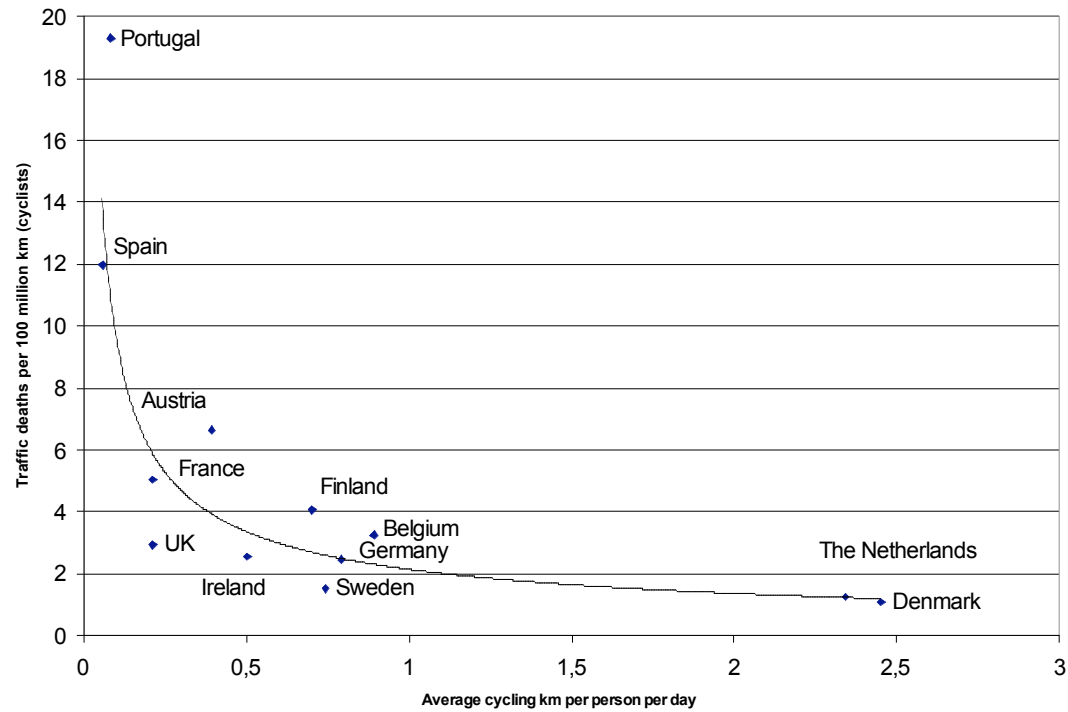
Walking and cycling rates to differ significantly between various countries. The table suggests that in most countries, especially in the USA, there is ample room for increasing physical activity levels by walking and cycling.

Supplement Material, Figure 1 Traffic deaths per 1 billion vehicle-km (2006).



Adapted from IRTAD-OECD <http://www.internationaltransportforum.org>.

Supplement Material, Figure 2 Relationship between average distance cycled per person per day and fatal traffic accident rate of cyclists (per 100 million km)



Adapted from IRTAD-OECD <http://www.internationaltransportforum.org>. and van Hout 2007.